

Organic bipolar disorder secondary to central neurocytoma resection: a case report

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Dear editor,

Central neurocytoma is a neuroepithelial tumor described by Hassoun in 1982, predominantly located in the midline at the level of the *septum pellucidum*, or in the lateral ventricle wall¹. They represent approximately 50% of intraventricular lesions in adults, and they are in total 0.25–0.5% of intracranial tumors². The origin of these tumors is not clear, but it seems that they come from bipotential neuronal and glial progenitor cells³. The mean age of the patients is 29 years, without presenting a predilection for either sex⁴. The typical clinical form of presentation is with symptoms of increased intracranial pressure, with symptoms and signs of normal pressure hydrocephalus⁵. The best treatment is surgical resection, this being curative, although tumor recurrence is relatively typical^{6,7}. A recent review on the possible neuropsychiatric complications after this type of intervention highlights the impairment in cognition, depressive symptoms and those of the anxiety spectrum⁸. On the other side, the onset of bipolar disorder occurs in a very small percentage of cases^{8,9}.

CASE REPORT

A 30-year-old woman with no organic history or toxicological habits of interest. The only notable family history was a paternal uncle diagnosed with paranoid schizophrenia. Neurology consultation due to gait apraxia and urinary incontinence. After pertinent complementary tests, he received the diagnosis of neurocytoma in the frontal horn and right ventricular body. She underwent neurosurgical surgery on two occasions, presenting intraoperative cerebral hemorrhage after the second intervention, affecting motor sequelae that had progressively remitted (she suffered hemiplegia and left hemiparesis with almost total recovery after rehabilitation) (Figure 1).

As a result of the resection carried out in the 2nd neurosurgical intervention, the mood decay began secondary to loss of functionality due to motor repercussions. It also describes cognitive complaints, loss of self-confidence and self-esteem. The patient was diagnosed with a moderate depressive episode, initiating treatment with duloxetine 60 mg, added to her anti-seizure treatment (100mg topiramate, 500mg levetiracetam and 100mg lamotrigine). She refers mood improvement with pharmacological readjustment and neurological rehabilitation.

At three months, she is referred to Neuropsychiatry consultations due to the persistence of depressive cognitions of the hopelessness, uneasiness, lack of interest and motivation in her activities, accompanied by an inability to carry out simple tasks of her daily life and free-floating anxiety. Likewise, it describes hyperphagic behaviors with anxiolytic purpose and a change is observed with respect to its previous characteristic pattern, having become more restless, impulsive and affectively unstable. The patient was diagnosed with

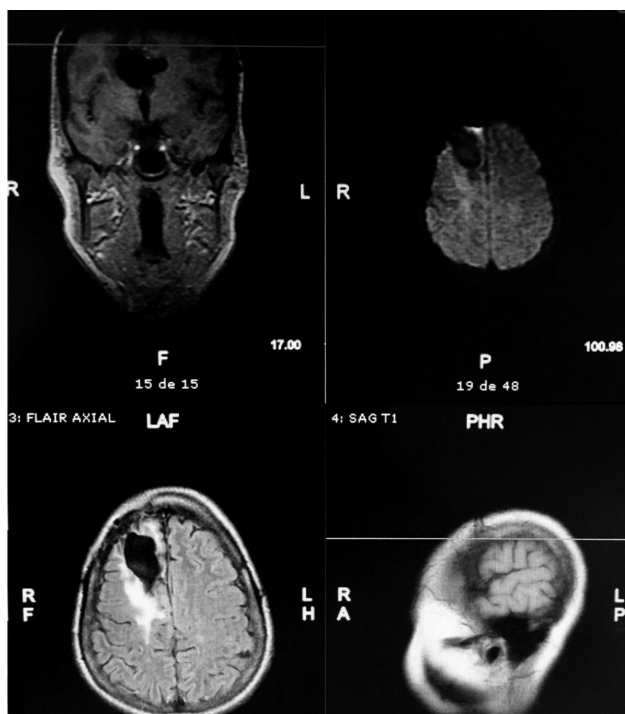


Figure 1

Craneoencephalic Magnetic Resonance. Coronal Sequence T1 (first image), T2X axial gradient (second image), axial FLAIR-T2 (third image) and sagittal T1 sequence (fourth image). Post-surgical changes are observed in the right front lobe with porencephalic cavity and peripheral gliosis and mild right lateral ventricular dilation.

a depressive episode with atypical characteristics, and it was decided to increase lamotrigine to 200mg as a mood stabilizer and the antidepressant treatment with duloxetine was maintained. With the medication adjustment, he refers to an improvement in the anxiety symptoms, although he still describes a remnant of mental instability.

At nine months he returned to consult for a new depressive episode. On this occasion, he refers to suffering from a morning worsening with increased anxiety symptoms, with significant tightness in the pit of the stomach, apathy, anger, anhedonia, insomnia and feelings of inability to face the day. After receiving a diagnosis of a major depressive episode, a change was made from duloxetine to desvenlafaxine up to a dose of 150mg / day and quetiapine 50mg was added at night. With the adjustment of the medication, he describes relative improvement, counting weeks in which he is "too well", with impulsivity and irritability, notably increased compared to his character base.

A year later, a period of continuous emotional oscillations began with a predominance of sadness, irritability, nervousness, impulsive reactions, anxious hyperphagia with bingeing on sweets and ideas of death. Given the cognitive sequelae of the patient secondary to the intervened neurocytoma, she presented amnesia for most of these episodes. Likewise, she presents difficulties in organizing tasks, such as planning domestic tasks, loss of orientation in habitual places, taking care of her own daughter, managing money, etc.

At two months, it begins with symptoms of a maniform tint consisting of hyperfluid language with decreased response latency, with tumultuous speech, difficult to interrupt from which ideas of megalomaniac tint are inferred without forming a systematized delusion, in the form of presumptions, divination and hyperactivity cerebral. Also, hyperthymic, expansive mood, with jocularity and a certain emotional lability. He does not present dysphoria or irritability, but there is disinhibition and increased activity. Describes hyperphagia, an anxiety component and global insomnia, with a lesser feeling of need. Given the apparent decompensation of her basic psychopathological picture in the form of a manic episode, the patient was diagnosed with Bipolar Disorder and a decision was made to enter a psychiatric hospitalization ward.

In the first days of her stay in Planta, she is expansive, hyperthymic with a speech centered on megalomaniacal ideas with delirious interpretations of magical content, as well as pseudocyesis. Treatment with valproic acid was started, the doses were adjusted upwards according to plasma levels, and treatment with desvenlafaxine was withdrawn. Eutimizing regimen is potentiated with aripiprazole 15 mg. With this change, progressive improvement is achieved with remission of the affective and psychotic symptoms.

At the cognitive level, he presents chronically and after neurosurgical complications, a predominance of attentional cognitive-functional impairment that has already been diagnosed, which has progressively improved with clinical affective improvement. Although attentional fluctuation and executive difficulties with a circumscribed appearance persist.

At the present time mental stability is appreciated, presenting euthymia lasting two years. Manifest symptoms have subsided and no depressive symptoms are observed at this time. Not psychotic semiology. No pathological anxiety. Pharmacologically preserved chronobiological rhythms. No active autolytic ideation, as well as passive ideas of death. No self or hetero-aggressive gestures, with preserved reality judgment. Presents apparent insight into the difficulties in the cognitive, emotional and functional sphere, with some minimization. Regarding planning, he uses routinization and task sequencing as a compensatory / facilitating strategy to alleviate deficits in his daily life.

After discharge, a neuropsychological evaluation was carried out and he was diagnosed with post-surgical cognitive-emotional sequelae with functional repercussions, attentional fluctuation and executive difficulties with a circumscribed appearance and verbal memory. A control MRI is performed where suspicious images of the rest or recurrence of the operated lesion are not observed. Post-surgical changes persist, with right frontoparietal craniotomy and disappearance of the underlying extra-axial collection.

DISCUSSION

Taking into account the medical pathology of the patient and the temporal evolution, we consider that the described picture may be due to the organic correlate, rather than to a primary mental illness. Therefore, given the phasic course and the hyperthymic symptoms, it would be an organic Bipolar Disorder according to the ICD-10¹⁰ criteria or a Bipolar Disorder due to a medical condition according to DSM-5¹¹. This complication is very infrequent, representing around 2% of the neuropsychiatric complications of this intervention¹².

Depressive and anxiety symptoms are the most typical neuropsychiatric sequelae in resections of this type of tumor, after cognitive symptoms⁸. Throughout the evolution of the condition, the exposed patient has presented different affective symptoms and free-floating anxiety with behavioral or functional alterations secondary to them, which at first could fit as typical sequelae of the intervention.

He has also presented cognitive symptoms on the one hand typical of the intervention and on the other parallel to the affective symptoms (partial improvement is described with the stabilizing and antidepressant treatment). The most

affective cognitive symptoms could be framed as a dys-executive syndrome, in which for the moment the memory would be absent¹³. It is estimated that it does not fit into a typical frontal syndrome, since neither the symptoms on the one hand and the anatomical correlate on the other (the frontal horn of the lateral ventricle is affected, not exactly the prefrontal area of the brain) would fit this hypothesis^{14,15}.

The hypothesis that we may be dealing with a primary mental disorder seems unlikely due to the temporal correlate, the absence of previous psychopathology and the absence of a family history of bipolar disorder^{16,17}. In addition, there are studies that link right brain damage with symptoms of mania^{9,18}.

In order to minimize the cognitive effect and not alter the antiepileptic regimen, treatment with valproic acid was chosen¹⁹. In order to minimize the risk of extrapyramidalism described with other antipsychotics, enhance the stabilizing regimen, and improve the antidepressant profile, the combination with aripiprazole was chosen²⁰. This was coupled with psychosocial interventions in order to improve the adaptation of the patient to society²¹.

CONCLUSION

This clinical case is presented, which in our opinion may be illustrative of an unusual neuropsychiatric complication for which additional research is needed to better understand the clinical characteristics, prognosis, and treatment.

Conflict of interests. The authors declare that they have no conflict of interest in relation to the material written in this article.

BIBLIOGRAPHY

1. Asa SL, Mete O. Hypothalamic Endocrine Tumors: An Update. *J Clin Med* [Internet]. 2019 Oct 20 [cited 2020 Apr 14];8(10):1741. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/31635149>
2. Sharma M, Deb P, Sharma S, Sarkar C. Neurocytoma: a comprehensive review. *Neurosurg Rev*. 2006;29(4):270.
3. Korshunov A, Sycheva R, Golanov A. Recurrent cytogenetic aberrations in central neurocytomas and their biological relevance. *Acta Neuropathol*. 2007;113(3):303–12.
4. Hassoun J, Söylemezoglu F, Gambarelli D, Figarella-Branger D, von Ammon K, Kleihues P. Central neurocytoma: a synopsis of clinical and histological features. *Brain Pathol*. 1993;3(3):297.
5. Beltrán-Marín M, Riaguas-Almenara A, Mazas-Artasona LV, Vela-Marín A, Marín-Cárdenas MA, Solanas-Álava S. Neurocitoma central: Hallazgos en tomografía computarizada y resonancia magnética. *Rev Neurol* [Internet]. 2009 Oct 1 [cited 2020 Apr 1];49(7):383–4. Available from: <https://www.neurologia.com/articulo/2009164>
6. Wang M, Zhou P, Zhang S, Liu X, Lv L, Wang Z, et al. Clinical Features, Treatment, and Long-term Outcomes of Central Neurocytoma: A 20-Year Experience at a Single Center. *World Neurosurg* [Internet]. 2018 Jan 1 [cited 2020 Apr 1];109:e59–66. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/28958923>
7. Leenstra J, Rodriguez F, Frechette C, Giannini C, Stafford S, Pollock B, et al. Central neurocytoma: management recommendations based on a 35-year experience. *Int J Radiat Oncol Biol Phys*. 2007;67(4):1145.
8. Keng A, Stewart DE, Sheehan KA. Examining the Neuropsychiatric Sequelae Postsurgical Resection of Adult Brain Tumors Through a Scoping Review [Internet]. *Psychosomatics*. Elsevier Inc.; 2020 [cited 2020 Apr 1]. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/32139084>
9. Ummar S, Kumar N, Ramanathan SA. Organic bipolar disorder: An unusual neuropsychiatric sequelae following right frontotemporal injury. *Indian J Psychol Med* [Internet]. 2016 [cited 2018 May 12];38(3):257–9. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/27335525>
10. OMS OM de la S. Trastornos Mentales y del Comportamiento (CIE 10). Criterios Diagnósticos de Investigación. / World Health Organization. International Statistical classification of Diseases and Related Health Problems. 10th ed. OMS, editor. Madrid; 1994.
11. APA. Asociación Americana de Psiquiatría. Manual diagnóstico y estadístico de los trastornos mentales (DSM-5ª). 5ª. Arlington V, editor. Madrid: Editorial Médica Panamericana; 2014. 663–667 p.
12. Koponen S, Taiminen T, Portin R, Himanen L, Isoniemi H, Heinonen H, et al. Axis I and II psychiatric disorders after traumatic brain injury: A 30-year follow-up study. *Am J Psychiatry* [Internet]. 2002 Aug [cited 2020 Apr 1];159(8):1315–21. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/12153823>
13. Luna-Lario P, Seijas-Gómez R, Tirapu-Ustárroz J, Hernáez-Goñi P, Mata-Pastor I. Estructura factorial del cuestionario disejecutivo en una muestra de población Española con daño cerebral adquirido y quejas de déficit

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- de memoria. *Rev Neurol* [Internet]. 2012 [cited 2020 Apr 1];55(11):641–50. Available from: <https://www.neurologia.com/articulo/2012549>
14. Pedrero-Pérez EJ, Ruiz-Sánchez de León JM. Quejas subjetivas de memoria, personalidad y sintomatología prefrontal en adultos jóvenes. *Rev Neurol* [Internet]. 2013 Oct 1 [cited 2020 Apr 1];57(7):289–96. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/24052439>
 15. Pedrero-Pérez EJ, De Ruiz-Sánchez León JM, Morales-Alonso S, Pedrero-Aguilar J, Fernández-Méndez LM. Sintomatología prefrontal en la vida diaria: Evaluación de cribado mediante el inventario de síntomas prefrontales abreviado (ISP-20). *Rev Neurol* [Internet]. 2015 Jan 1 [cited 2020 Apr 1];60(9):385–93. Available from: <https://www.neurologia.com/articulo/2014545>
 16. Harrison PJ, Geddes JR, Tunbridge EM. The Emerging Neurobiology of Bipolar Disorder. Vol. 41, *Trends in Neurosciences*. Elsevier Ltd; 2018. p. 18–30.
 17. Frías Á, Palma C, Farriols N, Salvador A. Characterizing offspring of bipolar parents: a review of the literature. *Actas Esp Psiquiatr* [Internet]. 2015 [cited 2020 Apr 14];43(6):221–34. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/26631305>
 18. Jorge RE, Arciniegas DB. Mood Disorders After TBI. Vol. 37, *Psychiatric Clinics of North America*. 2014. p. 13–29.
 19. Pérez-Ceballos MA, Vega-Gil N, Sánchez MB, Armijo JA. Use of antiepileptic drugs in bipolar disorder [Internet]. Vol. 34, *Actas Espanolas de Psiquiatria*. 2006 [cited 2020 Apr 14]. p. 55–64. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/16525906>
 20. Vieta E, Franco C. Advances in the treatment of mania: aripiprazole. *Actas Esp Psiquiatr*. 2008;36(3):158–64.
 21. Lolic M, Vázquez GH, Alvarez LM, Tamayo JM. Psychosocial interventions in bipolar disorder: a review. *Actas Esp Psiquiatr* [Internet]. 2012 [cited 2020 Apr 14];40(2):84–92. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/22508073>